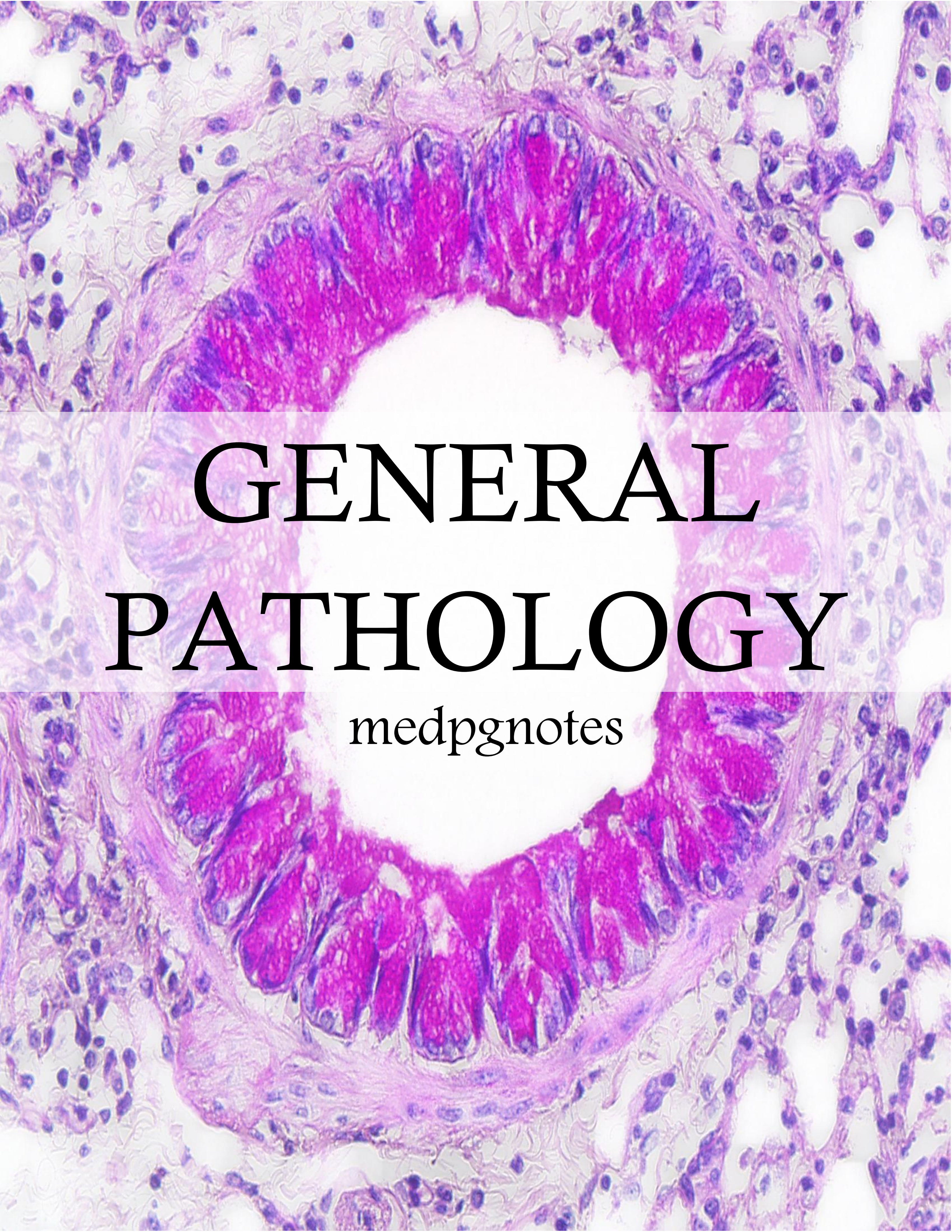

GENERAL PATHOLOGY

medpgnotes

GENERAL PATHOLOGY

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KEY TO THIS DOCUMENT

Text in normal font – Must read point. Asked in any previous medical entrance examinations

Text in bold font – Point from Harrison's text book of internal medicine 18th edition

Text in italic font – Can be read if you are thorough with above two

GENERAL FEATURES OF PATHOLOGY

FEATURES OF CELL INJURY

MC cause of Cell injury	Hypoxia
Feature of reversible cell injury	Myelin figures
Earliest tissue change following cell injury	Neutrophilia
Micropuncture of cell membrane with a needle. Repair occurs by	Linear movement of proteins
Reperfusion injury is caused by	Superoxide ion
Reperfusion injury is associated with elevation of	*Creatine kinase*
NOT a natural response to injury	Anabolism
Earliest change in reversible cell injury	Hydropic change
Electron microscopic finding of reversible cell injury	Flocculent densities in mitochondria
Reversible cell injury	Diminished generation of ATP, formation of blebs in plasma membrane, detachment of ribosomes from granular endoplasmic reticulum
Irreversible cell injury is seen in	Apoptosis
Irreversible cell injury is associated with	Accumulation of calcium in mitochondria
Most pathognomic sign of irreversible cell injury	Amorphous densities in mitochondria
Oncocytes are modified form of	Mitochondria
Hemorrhagic infarct	Lung
Red infarct	*Ovary, lung, small bowel*
Hemorrhagic infarction is seen in	Venous thrombosis, Thrombosis, Embolism
White infarcts are NOT seen in	Lung, Liver

HYPOXIA

Earliest indication of Hypoxia	Change in heart rate
First cellular change in hypoxia	Decreased oxidative phosphorylation in mitochondria

AGEING

Earliest change of ageing in cartilage	inability to regenerate articular cartilage
Cell aging is associated with	Decreased number of mitochondria, glycosylation of DNA, glycosylation of RNA, shortened telomere
Theory behind ageing process	Free radical theory
Ageing is due to	Free radical injury
Elderly occurrence of cancer is due to	*Increased telomerase activity*
Hormone increases as ageing occurs	*Cortisol*
Liver spot is associated with	**Ageing**
Replicative senescence is also known as	**Hay flick limit**
Progeria is due to mutation in	*Lamin A*
Werner syndrome	**Progeria of adult**

Hutchinson Gilford syndrome	**Progeria of children**
Barthel index for	**Assessment of ageing**
NOT associated with increased ageing	Increased superoxide dismutase
NOT compatible with ageing	*Decreased cross linkage*

NECROSIS

NOT a morphologic feature of necrosis	Hyperchromasia
NOT a feature of necrosis	*Cell shrinkage*
Type of Necrosis in hypoxic brain	Liquefactive necrosis
Liquefactive Necrosis	Brain
Pyogenic infection and brain infarction are associated with	Liquefactive necrosis
MC Type of Necrosis	Coagulative Necrosis
Coagulative necrosis is seen in	Myocardial infarction, thermal injury, tuberculosis
Coagulative Necrosis NOT seen in	Brain
Casseous necrosis	Tuberculosis , histoplasmosis
Casseous necrosis NOT seen in	Leprosy, CMV, Wegener granulomatosis
Fat necrosis	Breast, Omentum, Retroperitoneal fat
Fibrinoid necrosis may be observed in	Malignant hypertension, polyarteritis nodosa, Aschoff nodule
Fibrinoid necrosis	*Henoch Schonlein purpura, immune vasculitis, preeclampsia, hyperacute transplant rejection*
Fibrinoid necrosis is NOT seen in	Diabetic glomerulosclerosis
Necrotic keratinocytes	Graft versus host reaction, erythema multiforme, lichen planus
Calcification in necrotic tissue	Dystrophic calcification
Chemotherapeutic drug can cause	Both necrosis and apoptosis

GENERAL FEATURES OF APOPTOSIS

Apoptosis is defined as	Programmed cell death
Apoptosis in isolated cell	Death
Examples of apoptosis	Menstrual cycle, Tumor necrosis, GVHD, Pathological atrophy
Programmed cell death	Apoptosis
Toll like receptors recognize bacterial products and stimulates immune response by	FADD ligand apoptosis
Apoptotic bodies	Cell membrane found within organelles
Organelle involved in apoptosis	Mitochondria
Cell death is due to	*Deposition of extracellular amyloid*
Apoptosis can occur by changes in hormone levels in ovarian cycle. When there is no fertilization of the ovum, the endometrial cells die because	*Involution of corpus luteum causes estradiol and progesterone levels to fall dramatically*
Capsase involved in extrinsic pathway	*Capsase 10*

NOT a feature of apoptosis	Increase in cell size
NOT a feature of apoptosis	Cellular swelling
NOT true about apoptosis	Inflammation
NOT true about apoptosis	*Passive process*
Internucleosomal cleavage of DNA	Apoptosis
Features of apoptosis	Cytoplasmic blebs, Nuclear fragmentation
Light microscopic feature of apoptosis	*Intact membrane*
Apoptosis initiation	Death receptors induce apoptosis when it is engaged by fas ligand system
Councilman bodies associated with	Apoptosis
Ladder pattern of DNA electrophoresis in apoptosis is caused by action of	Endonuclease
In situ DNA nick end labeling can quantitate	Fraction of cells in apoptotic pathway
Characteristic feature of apoptosis on light microscopy	Nuclear compaction

APOPTOTIC AND ANTI APOPTOTIC PROTEIN

CD 95 major role in	Extrinsic apoptosis
Capsases are involved in	Apoptosis
Most important Executionary Capsase	Capsase 3
Capsases associated with	Organogenesis/embryogenesis
Increased p53 levels induce	Apoptosis
Cytosolic cytochrome c plays an important role in	Apoptosis
Leads to apoptosis	Glucocorticoid
Annexin V is a marker of	Apoptosis
Apaf is activated by	Cytochrome c
Which inhibits apoptosis in Memory B cells	Nerve growth factor
Memory cell in immune system are long lived and escape apoptosis because	Nerve growth factor
Anti apoptotic protein	Bcl 2, Bcl x
Gene inhibiting apoptosis	Bcl2

CALCIFICATION

Calcification begins in Mitochondria in all organs EXCEPT KIDNEY	Basement Membrane
Calcification of soft tissues without disturbance of calcium metabolism	Dystrophic calcification
Dystrophic calcification	Calcification in dead tissue
Dystrophic calcification is seen in	Atheromatous plaque
Psammoma bodies	Dystrophic calcification, Papillary Ca Thyroid, Serous Cystadenocarcinoma Ovary, Meningioma
MC Site of Metastatic Calcification	Lung
Metastatic calcification	Kidney
Metastatic calcification occur when calcium is in	Alkaline pH
Metastatic calcification starts in	Mitochondria
Metastatic calcification is commonly seen in	Renal tubules
Metastatic calcification	Mitochondria involves earliest

Common sites of metastatic calcification	Lung, kidney, gastric mucosa
NOT a common site of metastatic calcification	Parathyroid
NOT a site of metastatic calcification	*Pancreas*
Heterotopic calcification	Ankylosing spondylitis, Forrestier's disease (diffuse idiopathic skeletal hyperostosis)

ATROPHY AND HYPERTROPHY

Histological feature of disuse atrophy	*Type II fibres are involved, associated with angular atrophy*
Increase in size of cells	Hypertrophy
Example for hypertrophy	*Right ventricular hypertrophy due to systemic hypertension*
Both hyperplasia and hypertrophy found in	Pregnant uterus

HYPERPLASIA AND METAPLASIA

Cellular response that results from reprogramming of stem cells	Metaplasia
Metaplasia is	Reversible change, Slow growth, Reverse back to normal with appropriate treatment, If persistent may induce cancer transformation
Replacement of one epithelium by other	Metaplasia
Metaplasia from	Stem cells
NOT a feature of metaplasia	*Loss of polarity*
NOT true about hyperplasia	Increase in size of cell

STEM CELLS

Stem cells	Found in yolk sac, Used in gene therapy, Found in peripheral circulation, Proliferation and self renewal, Differentiation, Dormant phase of cell cycle
Example for multipotent stem cell	**Hematopoietic stem cells**
Helps in self renewal in hematopoietic stem cells	**Bmi1**
Oligopotent stem cells	**Neural stem cells**
Unipotent stem cells	**Spermatological stem cells**
Totipotency	Ability to form whole organism
NOT true about stem cell	Developmental elasticity
NOT a labile cell	Hepatocyte
Permanent cell	*Neuron*
Options of multiplicity of stem cell differentiation	*Developmental plasticity*
When stem cell transforms to other tissues, the process is known as	*Trans differentiation*

FIXATIVES AND STAINS

MC Fixative in Histological specimens	Formaldehyde
Commonly used fixative in diagnostic pathology	Formaldehyde
MC Fixative for Light Microscopy	10% Neutral Buffer Formalin
MC Fixative for Electron Microscopy	Glutaraldehyde
Negative staining in electron microscope	Osmium compounds
Best fixative for cytological studies	95% ethanol
Zenker's fixative	Mercury
Bouin fixative	Picric acid
Carnoy fixative	Lymph nodes in radical resection
Lipid in tissue detected by	Oil red O, Sudan III, Sudan black
Splenic macrophages in Gaucher's disease differ from those in ceroid histiocytosis by staining positive for	Lipid
PAS does NOT stain	Lipid
PAS does NOT stain	Basement membrane of bacteria
Colour of carcinoma is due to	Lipochrome
Light brown perinuclear pigment seen of staining of cardiac muscle fibre in grossly normal appearing heart of 83 old man at autopsy	Lipochrome
Non Endogenous pigment	Anthracotic Pigment
Brown atrophy is due to	Lipofuscin
Hemosiderin is stained by	Prussian blue
25 year renal transplant recipient died of meningitis. On autopsy, gelatinous exudates with cystic masses round encapsulated organism, stain	Prussian blue
Reticulocytes are stained with	Brilliant cresyl blue
Acridine orange is a fluorescent dye used to bind	DNA and RNA
Frozen section biopsy NOT used for	Amyloid
Tissue block specimen preserved for	10 years
Sebaceous gland ca-stain used	Oil red
Stain used for fungus	Silver methenamine
Silver methenamine stain is	Green
Hall's stain for	Bilirubin
Reticulin stain for	Type III collagen
Verhoeff , Van gieson stain	Elastic fibres
Luxol fast blue stain	Myelin
Ulex europaeus	Endothelium
Mucicarmine	Epithelial mucin
Von Kossa stain	Calcium
Method of Identification of Sentinel node during Breast Surgery	Isosulfan Blue Dye
Basement membrane in renal biopsy is stained by	**Jones-methenamine**
Collagen in renal biopsy is stained by	**Masson's trichrome**
Diastase removes	Glycogen
Glycogen is associated with	PAS reaction

PIGMENT

Pigment in liver is caused by	Lipofuscin, Pseudomelanin, Wilson's disease, Malarial pigment, Bile pigment
Wear and tear pigment in body refers to	Lipochrome
MC exogenous pigment is	*Carbon*
Brown atrophy is associated with	*Lipofuscin*

BACTERICIDAL SYSTEM

Most effective bactericidal system within phagocytes	Reactive oxygen metabolite mediated
Most important in bactericidal activity	*H2O2 – MPO – halide system*

HYDROGEN PEROXIDASE

Enzyme associated with formation of H2O2	Oxidase
Enzymes associated with breakdown of H2O2	Catalase, Peroxidase

OXIDATIVE STRESS

Enzymes reducing oxidative stress	Superoxide dismutase, Catalase, Glutathione peroxidase, Ceruloplasmin
Antioxidants	Vitamin C, Selenium, Glutathione peroxidase, Vitamin E
Does NOT handle oxidative damage in lens	Vitamin A
Does NOT reduce oxidative stress	Xanthine oxidase

FREE RADICAL

Free radicals involved in cell injury	*Superoxide anion, hydrogen peroxide, hydroxyl radical, peroxynitrite*
Enzyme contributing in generation of free oxygen radicals with in neutrophils for killing intracellular bacteria	Fenton's reaction, NADPH oxidase
Peroxisomal free radical scavenger	Superoxide dismutase, Glutathione peroxidase, Catalase
Pigment involved in free radical injury	Lipofuscin
Free radical scavengers	Vitamin A,C,E, Glutathione
Absorption of radiant energy can result in cell injury by causing hydrolysis of water. This type of injury is protected by	*Glutathione peroxidase*
Enzyme that protects brain from free radical injury	Superoxide dismutase
Fenton reaction leads to free radical generation when	Ferrous ions are converted into ferric ions
In genetic deficiency of MPO, increased susceptibility to infection is due to	Inability to produce hydroxyl-halide radicals

Glutathione reductase	Free radical scavenger
Free radicals are NOT generated by	Superoxide dismutase
NOT a free radical scavenger	Phosphorylase

NADPH OXIDASE

Reactive oxygen intermediates are released by	NADPH oxidase
Oxygen dependent killing done through	NADPH oxidase
Enzyme responsible for respiratory burst reaction	NADPH oxidase
Reactive oxygen intermediates are released by	NADPH oxidase
Enzyme catalyzing formation of Superoxide ion	NADPH oxidase
NADPH oxidase helps in formation of	Superoxide ion

BASEMENT MEMBRANE

Most Abundant Glycoprotein in Basement Membrane	Laminin
Laminin is found in	Basement membrane
Components of basement membrane	Nidogen, laminin, entactin
Type of collagen in basement membrane of kidney	Type IV
Collagen and basement membrane degraded by	Zinc dependent Matrix metalloproteinases
Function of Matrix Metalloproteinases	Degrade Collagen

INFLAMMATION

INFLAMMATORY MEDIATORS

Most potent mediator of inflammatory response	*Cytokine*
Cytokine	Include interleukin, Polypeptide (complex protein)
Cytokines	Polypeptide, Have autocrine and paracrine effect, Involve in leukocytic movements, Does NOT take part in intrinsic enzymatic reaction
Pyrogenic cytokine	IL-1, IL-6, TNF, Ciliary neurotrophic factor, IFN - alpha
Febrile response in CNS is mediated by	IL-1, interferon, tumor necrosis factor, bacterial toxin
Does NOT cause fever	IL-18
Major site for biosynthesis of cytokines	Keratinocyte
Cytokine secreted in sepsis and systemic inflammatory response syndrome	Neutrophils, platelets
COX II is induced by cytokines at the sites of	Inflammation
NOT a proinflammatory cytokine	IL-2
Interleukins are produced by	*Lymphocytes*
Interleukins function as	*Growth factors*
Fever is due to	IL-1
Interleukin secreted by macrophage stimulating lymphocytes	IL1

Inflammatory mediator of generalized systemic inflammation	IL1
IL 1 cause	Increased leukocyte adherence, Fibroblast proliferation, Increased collagen synthesis, T lymphocyte activation
IL-1 is synthesized by	Monocytes, Macrophages, Fibroblast
IL-1 produced by	T Lymphocyte activation, Macrophage
Endogenous Pyrogen	IL 1
Il-2 produced by	T-cells
Interleukin II is secreted by	T lymphocyte
Interleukin characteristically produced in TH1 response	IL2
IL 2 was previously called as	T cell growth factor
TH1 produces	IL-2
IL-2 antagonists	Tacrolimus, Sirolimus, Cyclosporine
IL-3 is associated with	*Hematopoiesis*
Source of IL-4	*T cells, mast cells*
Biological role of IL-4	*B cell proliferation, switching to IgE, TH2 differentiation*
Eosinophil production is increased by	IL-5
Paraneoplastic eosinophilia by	**IL-5**
TH2 produces	IL-5, IL-6
Myeloma cells are dependent on	IL-6
Granulocytosis and thrombocytopenia by	**IL-6**
IL 7 produced by	Stromal cells
Antigen gene rearrangement begins under the influence of	*IL-7*
Example of CXC or alpha chemokine	Interleukin 8
Chemotactic for neutrophils	*IL-8*
Recruitment of neutrophils	*IL-17*
Receptor for IL-8	**CXCR1, CXCR2**
CC beta	Eotaxin
CCL 11	**Eotaxin**
XCR1	**Lymphotactin**
Interferon	Inhibit viral replication in cells, Species specific, Reacts with cells and the affected cell then becomes resistant to number of different virus
Function of interferon	*Renders cell refractory to viral infection*
Major source of interferon alpha	*Macrophage*
Major source of interferon beta	*Fibroblasts*
Function of interferon alpha and beta	*Antiviral*
Major source of interferon gamma	*T cells, NK cells*
Interferon gamma is a	*Glycoprotein*
Biological effect of interferon gamma	*Activates macrophages, TH1 differentiation*
IFN gamma producing cells are measured by	*T spot*
Source of TNF alpha	*Macrophages, T cells*
Biological effects of TNF alpha	*Cell activation, fever, cachexia, antitumor*
Source of TNF beta	*T cells*
Biological function of TNF beta	*PMN activation*

IFN gamma is secreted by	T helper cells 1
Histamine is abundantly present in	Mast cells
Mesangial cells are contracted by	Histamine
Histamine	Mast cells, increases gastric acid secretion, related to arousal and blood pressure
Histamine cause	Vasodilatation
Histamine is formed by	Carboxylation of histidine
Histamine	Found in mast cells, Increases gastric secretion, Related to arousal and blood pressure
Flare in triple response is mediated by	Axon reflex
Lewis triple response is mediated by	Histamine
In Lewis triple response, Redness when skin is scratched with pinpointed object is seen due to	Histamine release due to local injury to mast cells
Acute phase reactants in inflammation	Fibrinogen and Haptoglobulin
Acute phase reactants	Fibrinogen, Plasminogen, Serum amyloid A protein, ESR, CRP, Haptoglobin
Acute phase reactants	*Ceruloplasmin, ferritin, alpha 1 antitrypsin, complement*
NOT an acute phase reactants	Albumin
C in C reactive protein stand for	Capsular polysaccharide of S.pneumonia
C reactive protein are	Beta 1 globulin, Non specific inflammatory protein
Half life of C reactive protein	18 hours
C reactive protein is	Increased in pneumococcal infection
C-reactive protein	Capsular polysaccharide, increased in pneumococcal infection
TGF α mediates	Tyrosine Phosphorylation
Neutralization of TGF beta affects	*Production of collagen*
ESR rise when there is increase in	Leucocytes
Source of serotonin	Platelets
Preformed mediator of inflammation	Serotonin
Toll like receptors recognize bacterial products and stimulates immune response by	Transcription of nuclear factor mediated by NFkB which recruits cytokines
Toll like receptors associated with virus	*TLR3*
Most important toll like receptor in humans	*TLR4*
Toll like receptor associated with mycoplasma	*TLR6*
Notch ligand associated with Increased Proliferation of Capillary Endothelial Sprouting	Delta like ligand 4
Inflammatory mediators NOT acting from cells	Kinin
Bradykinin	Pain, vasodilatation, bronchoconstriction
Bradykinin is for	Pain, vasodilatation and increased vascular permeability
Bradykinin in acute inflammation causes	Pain at the sign of inflammation
Causes edema and increased vascular permeability	*Bradykinin*
Drug inhibiting metabolic inactivation of bradykinin	*Captopril*
NOT a mediator of inflammation	Myeloperoxidase
MPO kills by	OCl-
Stabilization of Newly formed fragile vessels by	Ang 1, Ang 2

Does NOT help in STABILIZATION of newly formed blood vessels	*VEGF*

HYDROSTATIC AND OSMOTIC PRESSURE

Delayed prolonged leakage following tissue injury is due to	Direct effect of injuring agent
Increased capillary permeability	Anaphylotoxin, 5 hydroxytryptamine, histamine
Favors filtration at the arteriolar end of capillary bed	Increase in hydrostatic pressure of capillaries
Edema is caused by	Increased hydrostatic pressure
Cause of edema	Decreased plasma protein concentration
Albumin is important factor in maintaining osmotic pressure has	Low molecular weight and high blood concentration
Major determinant of colloid osmotic pressure in humans is	Albumin
Oncotic pressure is measured by	Osmol
Main function of Plasma oncotic pressure	Prevention of leakage from blood to tissues

GENERAL FEATURES OF INFLAMMATION

MC cause of Inflammation	Infection
MC type of Inflammation	Catarrhal
Signs of inflammation described by	Celsus
Rubor in inflammation is due to	Dilatation of arterioles
Pain in inflammation is mediated by	Bradykinin
Fifth cardinal feature of inflammation	*Loss of function*
Sequence of Inflammation	Initial Vasoconstriction, Vasodilatation, Hyperpermeability, Stasis
Cloudy swelling is	Reversible
Endothelium leukocyte interaction during inflammation is mediated by	Integrin - firm adhesion, Selectin - rolling and loose adhesion
Immigration of leucocytes through the vessel wall to site of inflammation	Diapedesis
Most Important molecule for Diapedesis	PECAM 1 or CD31
Leucocyte adhesion molecule involved in transmigration	*ICAM 1*
NOT true about inflammation	PCAM- adhesion
Inflammatory response is absent when Neutrophil count	< 200/mm3
Exudate	More protein, More specific gravity
Which is a Peripheral Cell membrane Protein	Cell Adhesion Molecule
NOT a cell adhesion molecule	Spectrin
Cathepsin D is associated with	*Invasiveness*

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME

Features of SIRS	WBC >12*10^9 or less than 4*10^9/L, RR>24/min, HR>90/min
SIRS criteria include	Tachycardia, leucocytosis, leucopenia
Systemic inflammatory response syndrome	Fever, Leucocytosis, hyperthermia, hypothermia
SIRS due to microbial cause	**Sepsis**
Autoinflammatory syndrome	**Group of febrile illness, mutation in inflammasome**
MC familial form of amyloidosis	Familial Mediterranean fever
Inherited auto inflammatory syndrome	Familial Mediterranean Fever
Familial Mediterranean fever	**Autosomal recessive**
Mutation in familial Mediterranean syndrome	**Pyrin**
Mutation in Muckle well's syndrome	**Cryopyrin**
Treatment of familial Mediterranean fever	**Oral colchicine daily**
Familial Hibernian fever is also known as	**TNF receptor associated periodic syndrome (TRAPS)**
Hyperimmunoglobinemia D with periodic fever syndrome	**Mutation in mevelonate kinase**

AUTOANTIGEN AND ASSOCIATED DISEASES

AUTOANTIGEN	**DISEASE**
Adenine nucleotide translocator	**Dilated cardiomyopathy**
Asialoglycoprotein receptor	**Autoimmune hyperlipidemia**
GM2 ganglioside	**Gullian Barre syndrome**
SOX 10	**Vitiligo**
Fibrillarin	**Scleroderma**
Gephyrin	**Paraneoplastic stiff person syndrome**
Recovertin	**Cancer associated retinopathy**
Synaptotagmin	**Lambert Eaton syndrome**
Yo protein	**Paraneoplastic cerebellar degeneration**

ACUTE INFLAMMATION

Most important role of bradykinin in acute inflammation	Increase In vascular permeability
In acute inflammation due to contraction of endothelial cell cytoskeleton	Early transient increase in permeability
In acute inflammation, endothelial RETRACTION leads to	Delayed prolonged increase in permeability
Immediate transient type of increase in vascular permeability in acute inflammation is seen in	*Venules*
Acute inflammation	Exudation, Vasodilatation, Margination
Characteristic feature of acute inflammation	Vasodilatation and increased vascular permeability
Hallmark of acute cytokine mediated inflammation	Endothelial cell expression of e- selectin
Calcium dependent ligand binding	*p-selectin*
Mediators of acute inflammation	Prostaglandins E2, Kallikrein, C3a, TNF-alpha, IL-1
Parasympathetic stimulation induces salivary acinar cells to release the	*Kallikrein*

protease	
NOT a mediator of acute inflammation	Angiotensin
NOT a change in acute inflammation	Decreased hydrostatic pressure
NOT an response to acute infection	Granuloma formation

CHRONIC INFLAMMATION

Chronic inflammation is characterized by	Lymphocytosis
Macrophages are derived from	Monocytes

CHRONIC GRANULOMATOUS DISEASE

Chronic granulomatous disease	**70% X linked, 30% recessive**
MC cause of Chronic Granulomatous disease in Children	Defect in NADPH, Oxidase enzyme complex of phagocytes
MC cause of chronic granulomatous disease in children	Defective H2O2 production
Nephrocalcinosis in a systemic granulomatous disease is due to	Overproduction of 1,25 dihydroxy vitamin D
Pathophysiology of Chronic Granulomatous disease	Decreased Oxidative burst
Defect in chronic granulomatous disease is due to failure to degrade	Chemoattractants
Chronic granulomatous disease characterized by	Abnormal bacterial phagocytosis
Chronic granulomatous disease	Associated with formation of multiple granuloma
Neutrophil defect associated with increased infections with catalase negative organism is associated with	*Chronic granulomatous disease*
Dihydrorodamine test is for	**Chronic granulomatous disease**
Nitroblue terazolium test for	**Chronic granulomatous disease**
Epithelioid like Giant cells	Brucellosis, Crohn’s disease, Leprosy
Epitheloid cell and multinucleated giant cells of granulomatous inflammation are derived from	Monocytes , macrophages
NOT an Epitheloid cell	Islet cells
Granulomatous inflammatory disease in NOT caused by	Mycoplasma
Granulomatous inflammation NOT seen in	AIDS
Epitheloid granulomatous lesions are NOT found in	Pneumocystis carnii

GRANULOMA

Most characteristic of granuloma	Epitheliod cell
Epitheloid granuloma is caused by	Helper T cells
Non caseating granuloma are seen in	Byssinosis, Hodgkin’s lymphoma, Tuberculosis
Durk’s Granuloma	Brain
NOT a bacterial infection	Pyogenic granuloma
NOT true about pyogenic granuloma	*Bleeds rarely*
Granuloma is NOT seen in	Amoeboma
Non caseating granuloma NOT seen in	Metastatic carcinoma lung
Granuloma formation NOT seen in	Candidiasis

COMPLEMENT SYSTEM

Complement is	Heat labile
Genes encoding complement are located	Adjacent to class I molecules
Intestinal epithelium synthesize	*C1*
Macrophage synthesize	*C2, C4*
Spleen synthesize	*C5, C8*
Liver synthesize	*C3, C6, C9*
Complement binds to	Antigen antibody complex
Functions of complement	Killing microorganisms, membrane leaks, increased vascular permeability
NOT true about complement	They are non specific immune response
Pathway involving direct activation of C1	*Lectin pathway*
Complement deficiency associated with high risk of SLE	*C1q*
MC Complement Deficiency	C2
Critical step in Complement system	Activation of C3
Acute phase reactant	C3
Most abundant complement in human	C3
Common link in classical and alternate pathway	C3
Stimulates bone marrow	C3b
C3b functions as	*Opsonin*
NOT a chemoattractant	C3
Factor present in final common pathway	C5
Anaphylotoxins	C3a, C5a
Most important mediator of chemotaxis	C5a
Endogenous chemoattractant	C5a
Complement playing role in arachidonate metabolism	*C5a*
C5-C8 deficiency is associated with	Septic pyogenic infection
Complement D, I, C2 associated with	**Pyogenic infections**
Activation of bacterial lysis	C5-9
Mediated through complement activation	Necrotizing vasculitis
Best denotes classical complement pathway activation in immune inflammatory condition	C2,C3,C4 decreased
Complement classical pathway is activated by	Immune complex
Complement components whose deficiency is associated with immune complex diseases	*C1q, C1rs,C2, C4*
Deficiency of C3 is associated with	*Pyogenic infections*
Terminal complement deficiency	Gonococcal arthritis
Complement H deficiency is associated with	**Hemolytic uremic syndrome**
After binding of complement and antibody on the surface of encapsulated bacteria, the process of polymorphonuclear leukocytes involve	Pseudopod extension
Test for complement	*CH50*

OPSONIZATION

Opsonin	Carbohydrate binding protein
Opsonisation occurs due to	Complement
Process of increasing ability for phagocytosis of foreign bodies	Opsonisation
Which process makes the bacteria tasty to macrophages	Opsonisation

PHAGOCYTOSIS

Phagocytosis	Metchnikoff
Size of particle ingested in phagocytosis	>0.5 microns
Most important function of epitheloid cells in tuberculosis	Phagocytosis
Phagocytosis	Amoeba and other protozoa make their living out of this, used to ingest particle >0.5 micron size, digestion occurs with phagolysosome
During phagocytosis, metabolic process called respiratory burst involves activation of	Oxidase
Phagocytes elaborate	Leukotrienes
Disorders of Phagocytosis	Lazy Leucocyte syndrome, Schwachmann Syndrome, Tuftsin deficiency
Nitroblue terazolium test	Phagocytosis
Phagocytic function is assessed by	Reduction of Nitroblue Tetrazolium test

CHEDIAK HIGASHI SYNDROME

Chediak higashi syndrome is due to	Failure of phagolysosome formation
Chediak Higashi syndrome is due to	*Decreased leucocyte function due to mutation affecting protein involved in lysosomal membrane traffic*
Chediak higashi syndrome	Neutropenia, Defective microbial killing, Presence of large granules in neutrophil, Immunodeficiency
Chediak higashi syndrome is due to defect in	Chemotaxis
NK cell hyporesponsiveness is seen in	**Chediak Higashi syndrome**
Large misshaped granules is associated with	**Chediak Higashi syndrome**

CHEMOTAXIS

Locomotion across chemical gradient is called	Chemotaxis
Main feature of chemotaxis	Unidirectional locomotion of neutrophils
Exogenous chemotactic agents	*Bacterial products*
Endogenous chemotactic agents	*C5a, LTB4, IL-8*
Binding of chemotactic agent to leucocyte	*Phospholipase C*

membrane receptors cause elevation of	

NEOPLASIA

CELL CYCLE

Sequence of Cell cycle	G0-G1-S-G2-M
Cell cycle	G0 G1 S G2 M, DNA synthesis occur in S phase, Cells divide in M phase
Phase determining length of cell cycle	*G1 (under control of p53)*
1st checkpoint in Cell cycle	G1S
Fixed time in cell cycle	S,G2,M
DNA replication occurs in	S phase
Cellular content of DNA is doubled in	S phase
Most radiosensitive phase of cell cycle	G2M
Phase of chemotherapeutic agent most resistant to chemotherapy	*Go*
First Cyclin to present in Cell cycle	Cyclin D
Initiation of DNA replication involves formation of	Cyclin E/cdk 2
Main mediator that propels cells beyond Prophase	Cyclin B/cdk1
Associated with G2M transition in cell cycle	Cyclin B
Why fetal cells continue to divide but terminally differentiated adult cells do not divide	Many cyclin inhibitor which prevent cell to enter in to S phase in adult

CAUSES OF NEOPLASIA

Oncogenic RNA virus	Hepatitis C virus
Oncogenic RNA virus	Human T cell Leukemia virus
LMP 1 gene plays a role in oncogenesis induced by	EBV
Effect of Oncogenic virus on affected cell is	Cell transformation and Lose property of contact inhibition
NOT a neoplastic virus	Cytomegalovirus
NOT a natural carcinogen	Benzidine
Hereditary factors are important in	Breast carcinoma, Retinoblastoma
Does NOT predispose to malignancy	Cri du chat Syndrome
Cri Du Chat syndrome	*Loss of 5p chromosome*
Polyvinylchloride	Angiosarcoma
UV-B	Skin cancer
Asbestos	Lung cancer
Aniline	Bladder cancer
Benzopyrine change to carcinogen in animal occurs due to	Epoxide formation, p53 activation, by inducing metabolism of CYP P450
Premalignant conditions	Down syndrome, Blount syndrome, Fanconi syndrome
Increased risk of cancer seen in	Chronic ulcerative colitis

NOT a premalignant condition	Fragile X syndrome
NOT associated with malignancy	Fragile X syndrome
Involved in tumor metastasis cascade	E cadhedrin

FEATURES OF NEOPLASIA

Number of cells to make a tumor	*10^9*
Malignant tumor of mesenchymal origin	*Sarcoma*
Hamartoma	Proliferation of native cells in tissue
Normal excessive tissue in normal site	Hamartoma
Normal tissue in abnormal location	Choristoma
Jigsaw puzzle pattern is seen in	Cylindroma
Autonomous hyperplasia	Fibromatosis
Metastasis in neoplasia does NOT to	Spleen
Lack of differentiation	Anaplasia
Earliest change of neoplastic transformation seen at microscopic level is	Dysplasia
Dysplasia refers to	Change in morphology
Reversible loss of polarity with abnormality in size and shape of cells in known as	Dysplasia
Phenotypic characteristics of neoplastic cell	*Dysregulated cell proliferation, failure to differentiate, loss of normal apoptotic pathway*
Angiogenesis	Formation of new blood vessels
Angiogenesis is NOT stimulated by	HGF
Pattern of growth in tumors	**Gompertzian pattern**
Warburg effect	*Tumor cells ferment glucose into lactose in presence of sufficient oxygen*
Essential for tumor metastasis	Angiogenesis
Histological benign lesion can be fatal if it	Transform to Carcinoma
Tumor associated hypoglycemia is due to	*IGF II*
RECIST criteria for	*Solid tumors*
NOT a malignant tumor	Fibromatosis
NOT a feature of malignant transformation of cultured cells	Increased requirement of growth factors
Sure sign of malignancy	Metastasis
Malignant tumor will show	Contrast pooling
NOT a histopathological finding of malignancy	No pleomorphism
NOT a non metastatic complication of malignancy	Cerebral cortical degeneration
Secondaries are NOT common in	Hand and feet bones
Migratory thrombophlebitis is seen in	Disseminated cancer
Migratory thrombophlebitis	Lung cancer, prostate cancer, pancreas cancer
Migratory thrombophlebitis is commonly associated with	Pancreatic carcinoma
Trousseau sign	Ca lung, stomach, pancreas
Trousseau sign NOT associated with	Liposarcoma
Migratory thrombophlebitis NOT associated with	Prostate cancer
Paraneoplastic syndrome	Cerebellar degeneration, amyotrophic lateral sclerosis, oposclonus myoclonus

Paraneoplastic sydrome is mostly associated with	Small cell carcinoma
Ames test	Simple bacterial test for mutagenic carcinogen
Most common carcinoma in females is of	Cervix
Round cell tumor	Neuroblastoma, Ewing's sarcoma, Non Hodgkin's lymphoma
Anticarcinogens are	Carotenoids
Important role of serum LDH	Tumor bulk
Oncological emergency	Spinal cord compression, SVC syndrome, tumor lysis syndrome
Acute emesis occurs within	**24 hours**
Nutrition in cancer cachexia is indicated when	**>10% of body weight**
Burnout syndrome	**Fatigue, disengagement, loss of self fulfillment seen in physicians**

PROTOONCOGENES AND TUMOR SUPPRESSOR GENES

Care taker gene	*Preserves genomic integrity*
Gate keeper gene	*Promotes differentiation eg p53*
Required for Normal growth	Proto oncogenes, Tumor suppressor gene, DNA repair genes
Oncogenesis	Proto oncogenes are activated by chromosomal translocation, Malignant transformation involves accumulation of mutations in protooncogenes and tumor suppressor genes, Point mutation of somatic cell, At G2M phase there is loss of inhibitors controlling cell cycle, Topoisomerase II cause breaks in strands, Decrease in telomerase activity cause antitumor effects
Oncogene	Are transduced from virus infected cell
Oncogene is a	Native component of DNA
Growth factor oncogene	Sis
MC abnormality of dominant oncogene in human tumors	RAS
RET oncogene	**Lymphoma**
WNT 1 oncogene	**Retinoblastoma**
MYB oncogene	**AML, CML, colorectal melanoma**
JUN oncogene	**Lung cancer**
AKT 1 oncogene	**Stomach**
AKT 2 oncogene	**Ovarian, breast, pancreas cancer**
Fos oncogene	**Osteosarcoma**
Met oncogene	**Osteocarcinoma, kidney cancer, glioma**
MET oncogene	**Hereditary papillary renal cancer**
PTEN	**Cowden syndrome**
Cowden syndrome is associated with	*Fibrocystic disease of breast and breast cancer, facial trichilemmoma, acral keratosis*
Gorlin syndrome	**PTCH 1**
First Tumor Suppressor gene	Rb
Tumor suppressor gene is important in	Retinoblastoma
Rb gene is active in	*Hypophosphorylated form*
CDKN2A	*Tumor suppressor gene coding for a*

	protein called p16 which acts at G1 to S checkpoint of cell cycle
Protooncogenes	Normally involved in cell cycle proliferation, Protooncogene on mutation cause cancer
Function of protooncogene	*Code for proteins that promote cell division*
Protoncogene erb B is related to	Breast carcinoma, Non small cell lung carcinoma, Ovarian carcinoma
Functions of protooncogene	Promotion of cell cycle progression, Inhibition of apoptosis, promotion of nuclear transcription
In mitogen activated protein kinase pathway, activation of RAS is counteracted by	GTPase activated protein
Small G protein related to	Product of ras protooncogene
MC nuclear transcription regulator in human tumors	MYC
C myc	Burkitt lymphoma
Myc	**Transcription factor**
NOT a function of protoncogene	Promotion of DNA repair
Promotion of DNA repair by	Tumor suppressor gene
Cancer Proliferation prevented by	Rb
Example for tumor suppressor gene	Rb,APC,p53
NOT a tumor suppressor gene	RAS
MC genetic mutation in carcinogenesis involve	P53
MC involver tumor suppressor gene in human	P53
Guardian of genome	P53
P53	Arrests cell cycle at G1 phase,53 kD protein
P53	Regulate certain genes involved in cell cycle regulation, Its increased levels can induce apoptosis, At the time of DNA injury following irradiation, its level increases and it acts to cause cell cycle arrest
NOT a cyclin dependent kinase inhibitor	P53
Increased susceptibility to breast ca associated with	P53
Mutation strongly associated with breast cancer	P53
P53 induce cell arrest at	G1 S phase
P53	53 kDa, Chromosome 17, Cause cell arrest in G1
P53 arrests cell cycle at	G1
NOT true about p53	Wild type p53 associated with childhood tumors
NOT true about p53	Its activity in cells decrease following UV radiation and stimulates cell cycle

MANAGEMENT OF NEOPLASIA

TNM classification of a malignant tumor signed as	Clinical staging
Her2/ neu receptor plays a role in	Predicting therapeutic response
DNA content and membrane antigen in tumor cells are measured by	Flow cytometry
Occult metastasis is detected by	PET scan
Dye used in chromoendoscopy for detection of cancer	Methylene blue
Sentinel node biopsy	Blue dye injected, Done to avoid inadvertent axillary lymphnode biopsy, Radioactive dye used

Selective action on hypoxic tumor cells	Mitomycin C
Beneficial in head and neck cancer	Concurrent chemotherapy

GENERAL FEATURES OF TUMOR MARKERS

Use of tumor markers	Screening of cancer, Follow up of a cancer patient especially for knowing about recurrence, For monitoring treatment of cancer
Cytokeratin is seen in	Carcinoma
56 year old chronic smoker, mass in bronchus resected. Most useful immunohistochemical marker	Cytokeratin
Follicular lymphoma	Bcl 2
Pancreatic neuroendocrine tumor	Gastrin
Malignant melanoma	S-100 sensitive, HMB 45 specific
Granulosa cell tumor	Inhibin
Seminoma	PLAP
Testicular teratoma	b-HCG
Lung cancer	Bombesin
Marker for ca pancreas	CA 19-9
Prostate cancer	PSA
Neuron specific enolase	**Small cell lung cancer, neuroblastoma**
LDH	**Lymphoma, Ewing's sarcoma**
Cytokeratin 7 positive	**Lung cancer, breast cancer, thyroid cancer, endometrial cancer, cervical cancer, pancreatic cancer, salivary gland cancer**
Cytokeratin 20 positive	**Colorectal carcinoma, merkel cell carcinoma**
Cytokeratin 7 positive, 20 positive	**Urothelial carcinoma, ovarian mucinous carcinoma, pancreatic adenocarcinoma, cholangiocarcinoma**
Both cytokertain negative	**HCC, RCC, prostate cancer, squamous lung cancer**
NOT a tumor marker	Beta 2 macroglobulin
NOT a tumor marker	Alpha 1 antitrypsin deficiency
NOT an example of tumor marker	Alpha hcg

CA-125

CA 125	Ca colon, breast, ovarian ca, bronchogenic ca, pancreatic ca, Pregnancy, endometriosis, ca endometrium, Ca prostate
CA 125 increased in	TB of genital organ, ovarian cancer, endometriosis
Ovarian cancer	CA 125

CEA

CEA is elevated in	Alcoholic cirrhosis, Ca colon, Emphysema
Colon cancer	CEA
CEA	Useful for screening of carcinoma colon, Helpful for follow up after resection, Levels decrease immediately after resection of tumor

CEA increased in	Lung cancer, Breast cancer, Colon cancer
CEA is NOT elevated in	Ulcerative colitis
Significance of reappearance of CEA after successful resection of tumor is that it spells	Beginning of the end

AFP

AFP structurally related to	Albumin
Maximum level of AFP	Fetal serum
Major source of AFP in fetal life	Yolk sac
Maximum level of fetal AFP	*Fetal serum*
Amniotic Fluid AFP is used for detection of	Neural Tube Defects
In pregnancy with increased MSAFP which should be done	USG
NOT true about alpha fetoprotein	Preoperative high value indicate worse prognosis
AFP raised in	Testicular tumor, Hepatoma, viral hepatitis
At 15 weeks of gestation, increased AFP in amniotic fluid	Intrauterine death
MSAFP increased in	Neural tube defects
AFP raised in	Hepatic ca, lung ca
Common cause of rise of AFP greater than 10 times the normal limit	Hepatocellular carcinoma
Alpha fetoprotein is increased in	Multiple gestation, maternal hematoma, abdominal wall defect
AFP is increased in	*Non seminomatous germ cell tumors*
Maternal serum alpha fetoprotein highest in	Gastrochisis
Highest maternal serum alpha fetoprotein	Gastrochisis
Testicular tumor marker	Alpha fetoprotein
Alpha fetoprotein is increased in	Hepatocellular carcinoma, Pancreatic carcinoma, Germ cells of testis
Liver carcinoma, Hepatoma is associated with increase in	AFP
Endodermal sinus tumor of testis is associated with increase in	AFP
MSAFP is decreased in	Down's syndrome
Decreased maternal serum AFP	Trisomy 21
AFP is NOT raised in	Kidney tumor
Screening with MSAFP do NOT detect	CTEV
Alpha fetoprotein is NOT increased in	Seminoma
Alpha fetoprotein is NOT increased in	Postmaturity
AFP is NOT seen in	Carcinoma colon
AFP is NOT seen in	Normal male
Alpha fetoprotein NOT raised	Dysgerminoma
Serum AFP is NOT increased in	Bladder carcinoma
AFP NOT increased in	Increased maternal weight

FEATURES OF TUMORS

Spontaneous regression	Malignant melanoma, Neuroblastoma
Surgically curable	Pheochromocytoma, Insulinoma, Appendicular carcinoid
Round cell tumor	Neuroblastoma, Ewing's sarcoma, Non hodgkin's lymphoma
Lynch II	Ca ovary, ca colon, ca endometrium
Lynch syndrome is associated with	Cancer colon, endometrium, ovary
Cancers associated with excess fat intake	Breast, colon, prostate
Erythropoietin secreting tumor	Cerebellar hemangioblastoma, Hepatoma, RCC, Fibromyoma of uterus